



Inhibitory vs Exhibitory neuron factors

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Abstract

This presentation will be focussing on how the core differences between our two major neuron types – Inhibitory and Exhibitory – play a major role in the balance which controls seizures. I will be discussing the original medical views on the matter, then how views changed once technology gave us greater insight into electrical activity within the brain – and finalise by relating current research to a very understandable analogy such as taste and sound to give the presentation a much wider audience scope.

Humans have between 90 and 170 different types of Neurons (depending on how you classify sub-categories) but they all actually fall into two categories – Inhibitory vs Exhibitory. This effectively means the signal that neuron sends out either generates more (exhibitory) or less (inhibitory) reactionary signals from the surrounding neurons. So basically, some neurons will generate more activity from the neurons around them – whereas others will cause the neurons around them to generate less activity. Prior Neurological studies into Epilepsy focussed mainly on the accelerant (Exhibitory) factor – that flaring was only caused by an increase in electrical activity. But we have found this is not correct. It is actually a balancing act that involves the complex processes of ion gating channels combined MRNA signals vs enzyme and protein regulation and charged ion volumes staying at a steady rate that does not activate the flaring process. And that's just a simplified view of the issue to say the least. A key factor we will be looking at is the role genetic signals play in ion gating channels that effect electrical conduction. Studies into SCN2A sodium regulation gene, CaMk11 enzyme, CNTN2 potassium regulators, PKD2 regulators, CRAC regulators, EAG2 potassium channels, MRNA Sodium channel regulators and Reline gene will be discussed as part of the presentation.

Biography

Paul Lang is CEO and Founder of ECA global foundation and in this presentation I have worked with our connections to focus recent studies into MRNA variations in both signalling and translation plus systematic but essential mechanisms for alterations and/or multiple variations of pre-post synaptic translations. We also looked at how several of the neurochemical factors of proteins such as calcium, magnesium and glycine played roles in the MRNA regulatory system and how mutations could widely range variations in signal translation then subsequent neurological activity and disorders such as Epilepsy – but also Autism, Parkinson's and others.